Pink Polycyclic Ulcerations on the Lower Back and Buttocks

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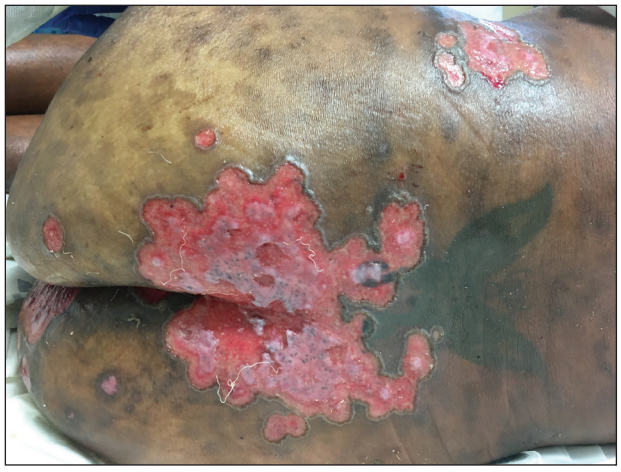

A 32-year-old woman with stage IV cutaneous T-cell lymphoma was admitted with pancytopenia and septic shock secondary to methicillin-susceptible *Staphylococcus aureus* bacteremia. Dermatology was consulted regarding sacral ulcerations. The lesions were asymptomatic and had been slowly enlarging over the course of 1 month. Physical examination revealed well-demarcated, pink, polycyclic ulcerations on the lower back and buttocks extending onto the perineum. There was no pain or tingling associated with the ulcerations. She denied a history of cold sore lesions on the lips or genitals. A skin biopsy was sent for tissue culture and histopathologic examination.

WHAT'S THE **DIAGNOSIS?**

a. candidiasis
b. contact dermatitis
c. cutaneous T-cell lymphoma
d. Epstein-Barr virus
e. herpes simplex virus

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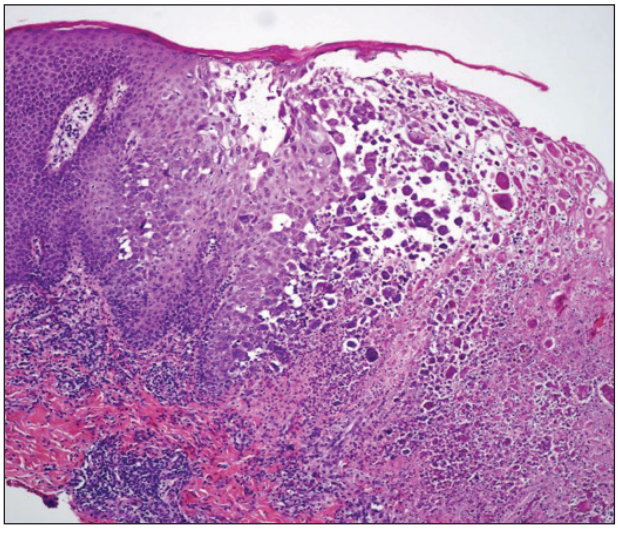
THE **DIAGNOSIS:** Herpes Simplex Virus

A skin biopsy was sent for tissue culture and was negative for mycobacterial, bacterial, and fungal growth. Histopathologic examination showed ballooning degeneration of keratinocytes with herpetic cytopathic effect consistent with herpetic ulceration (Figure). A swab of the lesion on the buttock was sent for human herpesvirus (HHV) and varicella-zoster virus nucleic acid testing, which was positive for HHV-2. She was started on oral valacyclovir 1000 mg twice daily for 10 days and then was continued on chronic suppression with 500 mg once daily. The patient's ulcerations healed slowly over the following few weeks.

Human herpesvirus 2 is the most common cause of genital ulcer disease and may present as chronic and recurrent ulcers in immunocompromised patients.[1] It usually is spread by sexual contact. Primary infection typically occurs in the cells of the dermis and epidermis. Two weeks after the primary infection, extragenital lesions can occur in the lumbosacral area on the buttocks, fingers, groin, or thighs, as seen in our patient,[2] which is a direct result of viral shedding and spread. Reactivation of HHV from the ganglia can occur with or without symptoms. Common locations for viral shedding in women are the cervix, vulva, and perianal areas.[3] Patients should be counseled to avoid sexual contact during recurrences.



Histopathologic examination showed ballooning degeneration of keratinocytes with herpetic cytopathic effect (H&E, original magnification ×10).

Cancer patients have a particularly increased risk for developing HHV-2 due to their limited cell-mediated immunity and exposure to immunosuppressive drugs.[4] Moreover, approximately 5% of immunocompromised patients develop resistance to antiviral therapy.[5] Although this phenomenon was not observed in our patient, identification of novel strategies to treat these new groups of patients will be essential.

The differential diagnosis includes perianal candidiasis, which is classified by erythematous plaques with satellite vesicles and pustules. Contact dermatitis is common in the buttock area and usually secondary to ingredients in cleansing wipes and topical treatments. It is defined by a well-demarcated, symmetric rash, which is more eczematous in nature. Cutaneous T-cell lymphoma was high in our differential given the patient's history of the disease. There are many variants, and tumor-stage disease may result in ulceration of the skin. Cutaneous T-cell lymphoma is differentiated by histology with immunophenotyping in conjunction with the clinical picture. Epstein-Barr virus (EBV) may cause genital ulcerations, which can be diagnosed with a positive EBV serology and detection of EBV by a polymerase chain reaction swab of the ulceration.

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